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Strabismology for which I contributed*

A estrabismologia para a qual contribuí

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ABSTRACT
Revision of changes in diagnostic and therapeutic methods, clinical and surgical of strabismus, mainly related to studies carried out by the author.

Keywords: Cycloplegia; Diagnostic methods; Viscosity; Magnetic Fields; Strabismus Surgery.

RESUMO
Revisão de mudanças de métodos diagnósticos e terapêuticos, clínicos e cirúrgicos dos estrabismos, principalmente relacionados a estudos protagonizados pelo autor.

Palavras-chave: Cicloplegia; Métodos diagnósticos; Viscosidade; Campos magnéticos; Cirurgias dos estrabismos.

RESUMEN
Revisión de cambios de métodos diagnósticos y terapéuticos, clínicos y quirúrgicos de los estrabismos, principalmente relacionados a estudios protagonizados por el autor.

Palabras Clave: Cycloplegy; Métodos Diagnósticos; Viscosidad; Campos Magnéticos; Cirurgía de Estrabismo.

Funding: No specific financial support was available for this study.
CEP Approval: Not applicable.
Disclosure of potential conflicts of interest: None of the authors have any potential conflict of interest to disclose.
Received on: Aug 23, 2018
Accepted on: Sep 05, 2018
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* Adapted from the major conference delivered at the end of the training course for specialists at the Strabos Institute in São Paulo on November 22, 2017 (The *strabismus I have experienced and the strabismus you will experience*, a theme proposed by the organizers of the event).
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How to cite: Bicas HEA. Strabismology for which I contributed. eOftalmo. 2018: 4(4): 186-209.
http://dx.doi.org/10.17545/eoftalmo/2018.0038

Vision, the sense on which ophthalmology and all ophthalmological care is founded, is one of the most fascinating subjects in human knowledge. The eyes, the organs at the start of vision, require a huge and complex set of variables to function, including coordination of their positions. It is, therefore, not surprising that the study of strabismus — the field of knowledge on eye coordination, which enables proper *binocular* vision — raises so many passions and embarrassment, either for the number of influencing factors or for the nature of its subtleties. Indeed, twelve external eye muscles innervated by three of the twelve pairs of cranial nerves and receiving commands from multiple structures of the central nervous system do not make this a simple subject. Although "each case is different" is a proverbial concept in Medicine as a whole, this maxim is particularly important for strabismus.

My academic life has aimed at analyzing the characteristics of strabismus thoroughly. Upon being invited to discuss this topic, I revisit what I consider the main considerations from my academic life.

BACKGROUND

As a result of the continued accumulation and renewal of scientific knowledge in its several fields, ophthalmology has also been divided into "subspecialties" to allow the necessary further developments to be reached to a greater extent. Strabismus was one of the first to become a separate field of study. Indeed, while in some ophthalmology fields interest groups dedicated to debate their topics were only beginning to be formed, there were already organizations being consolidated to study strabismus and its correlations: the *Consilium Europaeum Strabismi Studio Deditum* (*CESSD*, 1961) — which became the European Strabismological Association in 1982[1] ---, the Japanese Association of Strabismus and Amblyopia (1964)[2] and the International Strabismological Association (1966)[2,3]. In the USA, an informal "Squint Club" (with meetings since 1955) preceded the American Association for Pediatric Ophthalmology (1974)[3]. In Latin America (preceded only by CESSD and JASA), the Latin American Council on Strabismus was formed (1966)[4] and inspired the formation of the Brazilian Strabismus Center (1967), the CBE, the first "specialty society" within Brazilian ophthalmology. The second one, the Brazilian Society of Contact Lenses and Cornea, would arise only in 1971, and the third one, the Brazilian Society of Ocular Plastic Surgery, in 1974 — when CBE was already in its ninth scientific conference.

All this association effervescence may have been influenced by the development of orthoptics, an area dedicated to strabismus diagnostic tests and non-surgical treatments. Indeed, as diagnostic procedures related to strabismus demanded a lot of time and became increasingly complex, they started being delegated to paramedical professionals in 1930, becoming particularly relevant after World War II (1939-45). In the 1960s, orthoptics reached a peak in quality (extensive and diversified methods and techniques for examination and clinical treatment) and quantity (a large number of professionals prepared to use these methods and techniques). To exemplify the prominence of orthoptics in topics related to strabismus, it is enough to mention that, of the founders of the Brazilian Strabismus Center, ten were ophthalmologists and *eleven* were orthoptists.

That was what might be called the "sensorial period" of strabismus studies, that is, the time of the emphasis on sensory (visual) processes and disorders related to positional inaccuracies of the visual axes (oculomotor deviations), either as causes or as consequences. Even in academic circles of basic science, the attention given to the studies of the physiology of vision at the time can be illustrated by the Nobel prizes in Physiology or Medicine in 1967 (Ragnar Granit, Haldan K. Hartline, and George Wald, "for their discoveries concerning the primary physiological and chemical visual processes in the eye") and 1981 (David H. Hubel and Torsten N. Wiesel, "for their discoveries concerning information processing in the visual system," published mainly between 1963 and 1967).

Thus, a good ophthalmology facility should have among its education and health care personnel at least one orthoptist and several instruments of orthoptics (the most used being the synoptophore) and pleoptics (stimulation techniques for the recovery of mono- and binocular vision). These were the conditions of the Department of Ophthalmology of the Ribeirão Preto Medical School of the University of São Paulo when I began my residency in ophthalmology, in 1963. Gilda Baptista Soares De Sordi was the orthoptist in charge of the examinations and treatment follow-up of strabismic patients. Doctors were responsible for surgeries. And, precisely in that year, Gilda got pregnant and had to go on maternity leave. To calm down the head of Department, enraged by the perspective of loss of quality in strabismic patient care, Gilda decided to teach

the first-year residents (me and one other colleague) the science and art of orthoptics, initiating us in the handling of the essential (at the time) but obscure synoptophore. Maybe without Guy, the blessed fruit of that pregnancy, my academic path would have been different: my first paper (still as a sixth-year student and department monitor) was on intraocular pressure; my first studies in the (biochemistry) laboratory discussed the composition of vitreous humor; and my doctoral thesis was on keratometry. The fact is that I was soon (in October 1964) hired as a lecturer ("instructor") at the department from which I had graduated and assigned to coordinate the activities of the Strabismus Division because of both my engagement in the subject (nobody else wanted it) and my inclination and pleasure in performing these activities. And I appreciate that this has been my academic history.

THE SENSORIAL PERIOD

Suppression used to be measured by a synoptophore according to its *extent* (different figures presented to each eye, to assess the resulting simultaneous binocular perception) as *foveal* if simultaneous binocular perception was present even when images were very small; as *macular*, if there was cortical neutralization of only one of them in very limited central areas, corresponding to the fovea of one of the eyes; *peripheral* when they were larger; or, finally, *absent* when the patient reported no simultaneous binocular visual perception. Suppression *depth* also used to be measured by a colored transparent filter used in front one of the eyes to reveal diplopia, while the patients fixed their gaze to a light, being greater (more intense suppression, worse binocularity) when the color of this transparent filter used was denser. In this *simultaneous perception* condition, the magnitude of the angle formed by the respective visual axes during fixation — that is, the *deviation* between them — was the "basic" measure of strabismus (or heterophoria). **"Retinal correspondence"** was considered *normal* (N.R.C.) if the "objective" measurement of the deviation coincided with the "subjective" measurement (the angle between the visual axes in which the patient reported simultaneous perception), or *abnormal* (A.R.C.), if these measures were different from each other. When detected, A.R.C. was classified by type (harmonious, unharmonious) and intensity ("depth"). Orthoptic and pleoptic therapies were then indicated to recover from those abnormal states of binocularity.

Once *simultaneous (binocular) perception* (considered the most elementary degree of binocularity) had been detected, binocular ***fusion*** was measured, especially its *amplitude*, i.e., fusional convergence and the several types of fusional divergence (horizontal, vertical, and torsional).

Finally, and still using a synoptophore, the "third" (highest) degree of binocularly, ***stereopsis***, was assessed by using binocularly fusible images with slight differences.

The arsenal of devices necessary to an ophthalmology office included an equipment to examine visual behavior through ***Worth's four-dot test***. Using two filters, one red (on the right eye) and one green (on the left eye), patients were asked what they saw on a panel with four lights arranged around an imaginary center: a left and a right red light (of the complementary color to the green filter, so they would not be seen by the left eye), a top green light (of the complementary color to the red filter, so it would not be seen by the right eye), and a bottom white light, visible only by the right eye (if the left was suppressed), only by the left eye (if the right was suppressed), or alternately. Finally, the bottom light (white) was perceived as red if the right eye was the "dominant" or "fixing" eye, or green if these conditions fit the left eye. (See comments on the "Arthur Jampolsky" section.)

Regarding treatment, the synoptophore had what were called *Haidinger's brushes*, the image of a "shadow" (produced by polarized light seen through polarizing filters) going through a circle at the frontal plane — a technique used to stimulate the fovea to treat *eccentric fixation* and amblyopia. With the same purpose, *euthyscopy* was used, which was a form of direct ophthalmoscopy with intense peripheral lighting, except for the central area, preserving it so this region would then be the only one functionally "available" to fixate optotypes in front of the eye that underwent this treatment. A variant with a similar principle was *pleoptic therapy with after-images*, using a bright flashing light to "blind" (ephemerally) the peripheral retina, but preserving the central regions of the retina. The type and magnitude of deviations (in the absence of binocular suppression) were assessed by *Hess-Lancaster screens*.

Although it was not a strict rule, for deviations of relatively large angles, surgeons preferred to operate one of the eyes and later complement the correction of the remaining deviation by operating the other eye. That was also the routine for associated horizontal and vertical deviations: one of the deviations was corrected first and the other was corrected on another date. Conjunctival openings were semilunar, distant from the limbus; muscles were sutured to the sclera using catgut 4-0 (later chromic catgut, and then 5-0). Scarring granulomas were common. After surgery, occlusion lenses with central holes were used to “induce” centralized fixation binocularly.

Reflecting the close relationship between strabismus care and the exercise of orthoptics, the USP Ribeirão Preto Medical School Orthoptics Course was created in 1967, but it was short-lived, being extinct shortly after my return from studies at the Institute of Ophthalmology of the University of London (1969-71).

In any case, and despite the massive influence of sensory aspects on the examination and treatment of strabismus, my study interests were aimed at the influence of accommodative stimuli on the horizontal oculomotor balance (leading to convergence). Thus, my early studies were on accommodation[5], convergence[6-8], and on the synkinetic relationship between these two functions[9-18]. These more specialized studies resulted in the proposal for a new clinical trial[19] and in the annual award from the Brazilian Society of Ophthalmology (the Adaga “August Lohnstein” Prize, 1970) to one of the articles[20].

The intimate influence of accommodation on the oculomotor balance required — as it still requires —knowing refractometry absolute values. In fact, it was a “golden rule” to prescribe total correction of hyperopia, mainly for esotropia, since some cases could be completely resolved by this procedure (“purely accommodative” esotropia); others could be resolved by additional correction lenses “to see near” (bifocals, for “hypo-accommodative” esotropia, with high AC/A ratios); and, finally, in cases in which these optical corrections failed to neutralize far and near deviations, surgery was indicated, but exclusively to fix the “nonaccommodative” component, that is, what remained after total hyperopia correction. Therefore, in order to assess total (or absolute) refractometry values, cycloplegia was induced by 1% atropine eyedrops three times a day (morning, afternoon, and evening) for three consecutive days *prior* to the exam. A curious note is that this usage was very well accepted, although it required a second visit to complete the (refractometry) examination and even though the induced cycloplegia persisted for about two weeks, hindering school and visual performance of the children (and adults) examined; and, lastly, although this practice could be dangerous, since patients or their caregivers were handed a bottle of eyedrops (5 ml) with a total content of atropine (50 mg) equivalent to four times the potentially lethal dose for a 10 kg child[21]. However, a more interesting fact is that, despite all these inconveniences, this procedure could be considered *insufficient* to produce *total* cycloplegia. Actually, in subsequent refractometry for control a few months after the total correction of hyperopia, it was not uncommon to find *greater* hyperopia values, justifying the belief that an even more rigorous cycloplegic procedure was necessary. For this reason, even an *eight*-day atropinization was proposed[22] (although, in this study, only five of 41 cases presented an additional difference of 0.75 D in hyperopia with eight-day atropinization, compared with three-day atropinization).

With this hypothesis of *insufficiency* of three-day atropinization to achieve *total* cycloplegia, I invited a Bolivian ophthalmologist who had already completed his specialization in Colombia, but wished to broaden it in Brazil, and had proven to be excellent in refractometry measurements, to participate in a study about this procedure. In this study, each of us independently registered the value of the refractometry conducted by the other (who was blinded to this value). Patients should use 1% atropine eyedrops, one drop in each eye three times a day for *ten* days, being re-examined in the 3rd, 5th, 7th, and 10th days. They were divided randomly into three groups and had a refractometry on day zero (before the atropin onset) about 60 to 90 minutes after instillation of an eyedrop. Group I received *five* drops of 1% cyclopentolate [2-(dimethylamino) ethyl 2-(1-hydroxycyclopentyl)-2-phenylacetate] in each eye (one every three minutes) + 0.05% hyoscine; Group II received *three* drops of 2% homatropine (bromide), one every three minutes; and Group III received *two* drops of 1% atropine (sulfate), one every five minutes. In data analysis, at the end of the study, the refractometries conducted by both examiners had significantly similar “double” measurements. And, to a certain disappointment regarding the hypothesis to be proved, the measurements made on the 3rd, 5th, 7th, and 10th days were also very similar to each other; the effect was not potentialized nor accumulated with continued doses. And, for an even greater surprise, the initial measurements in any of the groups were virtually “identical” to those after extended atropinization. That is, regardless of the drug used, the same cycloplegia

was achieved, not increasing with classical atropinization (three days) nor with continued doses[22,23]. These results led to the proposed change from the classical method of cycloplegia for strabismus to an *immediate* (in-office) method, but still suggesting the preferred use of atropine[23] and that, on this day, it be instilled three times[24]. Cyclopentolate would only replace atropine in cycloplegia later. A young man from a South American country could not dare more than this.

ARTHUR JAMPOLSKY

He was not the only one to influence the development of strabismology in Latin America, but he is the one who developed it the most in mode, quality, and intensity, and is rightly recognized for it.

Jampolsky created and modeled the Smith-Kettlewell Institute of Visual Sciences (now Smith-Kettlewell Eye Research Institute) in San Francisco, a center for advanced research in the field of ocular motility that soon became the reference for whoever wished to delve into the *science* (more than in the *art*) related to strabismus. With a great ability to explain and convince, Jampolsky transformed practices and ways of thinking and acting. With brief, logical, and even tautological arguments, he taught: "If someone plans a single surgical correction, he (or she) may have to perform a second one. But if the initial plan is to perform two, he (or she) will perform at least two; and perhaps will have to perform three (or more)." "If someone plans a correction of the horizontal deviation and not of the vertical one, the remaining (and expected) vertical deviation will help decompensate the horizontal one again." And there are other quotes as these ones, to conceptualize that strabismus surgical planning should contemplate full correction of *quantity* and *type* at once. The "end of orthoptics" in Latin America is attributed to him due to quotes such as: "Why use Worth's four-dot test? The type of strabismus presented already predicts the result of the exam" (an obvious conclusion, after a simple reflection on the purpose of the test). "The doctor should examine the patient, and not delegate it to others" (learning by doing). "It is unfair to say we do not have a synoptophore in the Smith-Kettlewell Institute of Visual Sciences (SKIVS); we do, in the museum" (to demystify the importance of this instrument and emphasize the teaching that tests should be performed with eye fixations to objects in the "real" space, in front of the patient). Two quotes from Argentinian physician Alberto Ciancia, who later became president of the International Strabismological Association, illustrate the situation back then quite well: "If Jampolsky is right, we will advance twenty years; if he is wrong, we will go back twenty years." And later: "Before Jampolsky, the study of strabismus was simply dreadful; after Jampolsky, it became dreadfully simple."

In the strabismology practiced at the time, a field in which technological advances were not prevalent, the famous Latin American ingenuity (to some extent stimulated by the lack of strict control standards for innovations and paradigm breaking) could be showing its merits and need for incentive. And it was precisely Jampolsky — who always held the studies presented in the Latin American Council on Strabismus (CLADE) conferences in high regard — who was one of the first to value studies conducted here and show respect for them. His mentions of Latin American strabismus studies in his superb conferences, and his participation in round tables of *other* world conferences, as well as the emphasis he gave to their latinamerican authors, were a gift to their self-esteem and confidence.

I particularly owe Jampolsky one of the highest honors I ever received. In 1972, A. Edward Maumenee (1913-98), head of the Wilmer Institute of the famous Johns Hopkins University (Baltimore, MD), asked Jampolsky whom should he invite to replace the very respected Gunter K. von Noorden (1928-2017), who had been the head of the Strabismus Division and was leaving for the Baylor College of Medicine (Houston, TX)[3]. Jampolsky referred me. I was then asked to teach a course on strabismus, so the faculty at the Wilmer Institute could give their opinion. After the course was over, Maumenee proposed a six-year contract to me (which I found too long, unsure whether my family would adapt to Baltimore) and mentioned that I should dedicate the first years to reorganizing the outpatient and surgical services, without a lot of time for research. I would have to resign from the university and be far from my family and affective roots in Brazil, which scared me because it seemed like a point of no return. I asked him some time to decide. From Baltimore, I headed to San Francisco to get to know the Smith-Kettlewell Institute of Visual Sciences and personally thank Jampolsky for this almost unbelievable referral. While we talked, he noticed my indecision and was generous: "Come to Smith-Kettlewell." I was surprised at this distinguished invitation: "To do what?" "Whatever you want." "For how long?" "As long as you want." "When?" "Whenever you want." There could not be a more enticing proposition, and that was how I made my decision. I went to SKIVS (1974-75). And I appreciate that this has been my history.

Thus began a new phase of my academic life, which coincided with a major shift in the way strabismology developed.

THE MECHANISTIC PERIOD

At the time when orthoptics was at its peak, studies regarding "forces," "elasticities," "tensions," "velocities," "accelerations," "work," "energy," "vectors," and "matrices" were being designed, influenced by orthoptics itself, with greater association to topics of dynamics and physics than to psychobiology[25-29] for which a mathematical language was required. These topics became preponderant in strabismology and shed a new light on it. More knowledge was being accrued regarding eye mechanics, in particular studies on forces acting on the oculomotor system and their effects. These forces will be presented separately herein although the dissociation of these component forces of the oculomotor balance is artificial and even contradictory because there is a close interaction between the so-called "active" forces (related to the innervational control of the system that, in turn, controls the position and movement of the eye) and "passive" forces (caused by the components of the periocular structures). For example, a specific muscle relaxation "allows" the eye to rotate in one direction, while its contraction rotates the eye in the opposite direction; however, because the muscle distends with relaxation, this "allowed" rotation is simultaneously restricted. But the fact that studies focus on either one of these forces than on both justifies the convenience of describing them separately.

A) Active forces

Jampolsky first visited Brazil in September 1971 (16th Brazilian Congress of Ophthalmology, Campinas). I recall his flattering comments about my presentation during a symposium on "vertical deviations," when I spoke about "Investigation and diagnosis of vertical deviations". It is possible that, on that occasion, I referred to my previous observations about the inconsistency of the classical Parks test, the gold standard for the diagnosis of the "muscle responsible" for a vertical deviation. But the praises were for a concept that introduced the presentation: that there were *four* eye elevator muscles (and not just the superior rectus muscle and the inferior oblique muscle) and *four* eye depressor muscles (and not just the inferior rectus muscle and the superior oblique muscle). The two additional muscles, in both cases, were the horizontal rectus muscles (medial and lateral). The observation did in fact confront the notions that prevailed regarding the actions of the extraocular muscles because they had been defined by the well-known diagrams of Márquez and Van der Hoeve[30,31] (Figure 1) and explicitly reinforced by the teachings of one of the most studied text books, which attributed a ***single*** horizontal action to the horizontal recti muscles: *"Droit externe. Il est abducteur et c'est là sa seule action." "Droit interne. Il est adducteur et c'est là sa seule action."*[30]

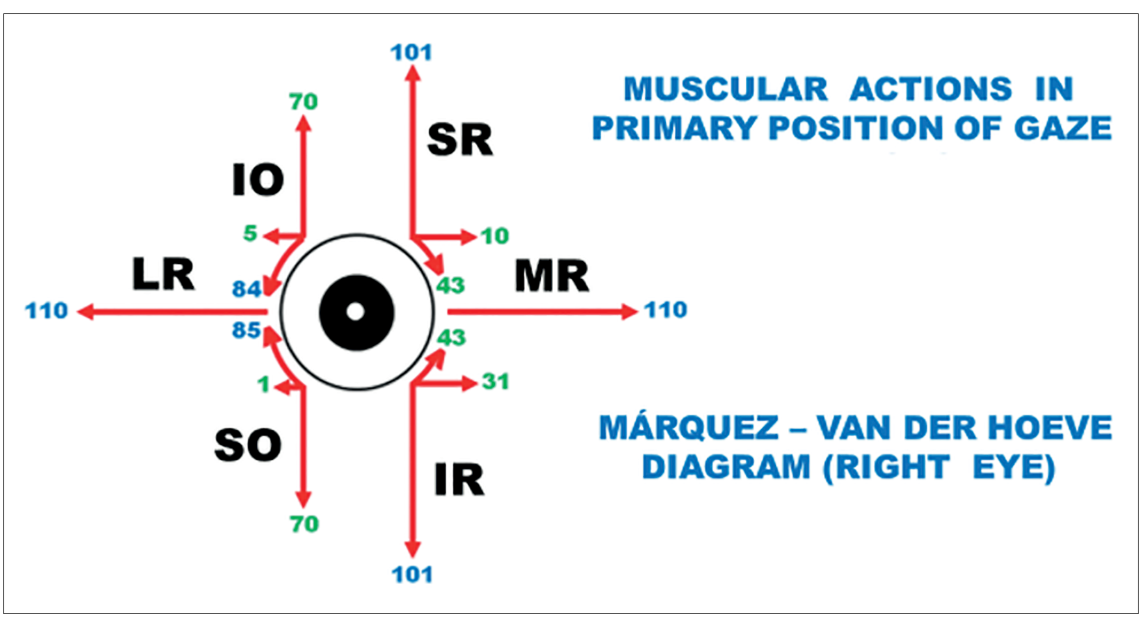


Figure 1. Márquez and van der Hoeve diagram showing the relative values of the horizontal (horizontal lines), vertical (vertical lines), and torsional (curved lines) actions of each extraocular muscle of the right eye in the primary position of gaze. The value of each muscle's predominant action is shown in blue. Note that for the horizontal rectus muscles, only horizontal action is considered.

The idea was not original; it revisited the dynamic approach to muscle actions of Castanera Pueyo[31] (as opposed to the static approach that prevailed at the time). This notion that the actions of an extraocular muscle vary according to the direction in which the main visual axis is pulled (in fixation) was based on the vectorial distribution of the force applied to the eye, originating from the contraction Krewson[27] had analyzed the vector components of the force of an extraocular muscle (E0M) at different points of fixation, but he limited his study to a single (horizontal) gaze direction. Boeder[28,29] took into consideration the total work performed by an EOM in different gaze positions, but he did not investigate the components in the fundamental planes. Therefore, I decided to cover all aspects, i.e., the different actions of each EOM (horizontal, vertical, and torsional) represented in a frontal plane, and how they could vary depending on the eye movement itself. This meant starting by studying the various systems that could define the ocular rotations[32] (it is interesting that a consensus on which a referential system should be adopted as standard has not yet been reached), and by performing a generic mathematical analysis of the eye rotations[33] to finally study the vector components of the force for each EOM in the different eye positions, represented in a frontal plane, with values up to 50 degrees in each cardinal direction, from the primary position of gaze[34]. The next step was the calculation of the length of each muscle (to determine the state of its contraction or relaxation) and of the work it performed (from the primary position of gaze at each position in space)[35]; the relationships between the arc of contact (between the muscle and the sclera), and the resulting rotation[36]. The last step was the study of the variation of the force exerted by an EOM for the visual axis to locate a given direction in space (starting from the primary position), which corresponds to an integration of the vectorial components of its action in each of the three fundamental planes of analysis (horizontal, vertical, and frontal, for the torsional actions)[37], and the respective performed work[38]. The publication of this series was preceded by a paper with a synthesis of such studies[39].

Concomitant to this study of *active* forces, which are the result of muscle action mediated by the oculomotor innervation, a concept of *passive* forces (relative to movement restriction)[40] and how they relate to each other to form a self-regulated system[41] was addressed (a study that was awarded the Barbosa da Luz Prize at the 19th Brazilian Congress of Ophthalmology in 1977) was also developed. The result of this study was a holistic concept about the complete integration of EOM functioning. In fact, on reexamination of Sherrington's law of reciprocal innervation, it became obvious that the action of a muscle, such as adduction through the *contraction* of the medial rectus, was only possible with the simultaneous *relaxation* of its antagonist, the lateral rectus muscle; in fact, in Type-I Duane syndrome, eye adduction is significantly decreased by the simultaneous contraction of the lateral rectus. Hence, it was postulated that the lateral rectus muscle was *also* an *acting* muscle (through relaxation) in *adduction*, i.e., adduction was primarily *initiated* by the medial rectus muscle, *aided* by synergistic muscles (the superior and inferior recti), and *allowed* by the relaxation of the respective opponents (the direct antagonist, lateral rectus muscle; and the indirect antagonists, obliques). Similarly, elevation was *initiated* by the superior rectus muscle and the inferior oblique muscle (being irrelevant for the concept whether the inferior oblique muscle has a primary or an adjuvant role in elevation), *aided* by the horizontal recti muscles (medial and lateral), and *allowed* by the relaxation of the respective antagonists (the inferior rectus and the superior oblique muscles). Thus, the EOMs involved in elevation are neither only the two classically accepted muscles (the superior rectus and the inferior oblique muscles), nor four (when including the horizontal recti muscles), but *all the six* muscles (now including the role of the inferior rectus and the superior oblique muscles in relaxation).

Another argument for this conception is that this interaction among the EOMs is not only casual but also indispensable for a *pure* rotation (in a single plane) to occur. Indeed, for a pure elevation, the superior rectus, the main elevator muscle, must be activated. However, this muscle produces, also, *adduction* and an *incycloduction*. The simultaneous relaxation of the inferior rectus, --- a depressor muscle, which contraction produces also, *adduction* and *excycloduction* --- would favor elevation, but it would be accompanied by *abduction,* which could eventually counteract adduction by the superior rectus, and by *incycloduction*, which is also promoted by the superior rectus. Conversely, participation of the obliques (contraction of the inferior oblique leading to elevation, abduction, and excycloduction and relaxation of the superior oblique leading to elevation, adduction, and excycloduction) would provide a combination of elevation and *excycloduction* (to counteract the incycloduction brought about by the vertical rectus muscles). The *horizontal* effect (abduction by the contraction of the inferior oblique muscle and adduction by the relaxation of the superior oblique muscle) would eventually be compensated by a supposed symmetry of action of such antagonist muscles. In any case, under circumstances wherein this supposed symmetry of the opposed horizontal actions (of the vertical recti and obliques) has to be admited (for example, in tertiary positions of gaze), *horizontal* imbalances (adduction or abduction) would also occur. Thus, as an

example, the prevailing participation of the horizontal action muscles is necessary to ensure that a rotation (for example, upwards and to the right) occurs. In other words, one muscle group would produce one type of action (the vertical rectus muscles generating *vertical* action but accompanied by unwanted horizontal and torsional actions, the annulment of which requires the *active* participation of the oblique muscles (to compensate for the undesired torsion associated) and of the horizontal recti muscles (to compensate for the undesired horizontal action asssociated).

Each *pair* of directly antagonistic muscles (vertical recti, horizontal recti, and oblique muscles) exhibits main actions in the primary position of gaze, which are respectively opposed, each action being *favored* by the contraction of one muscle and relaxation of the other according to Sherrington's law. Hence, the horizontal action is potentiated by the horizontal recti, the vertical action by the vertical recti, and the torsional action by the obliques. In the case of the vertical recti and oblique muscles, the torsional action of the former and the vertical action of the latter, although significantly reduced relatively to the main actions (vertical and torsional actions, respectively), are also potentiated by Sherrington's law, and this is the reason why these four muscles are called "cyclovertical" muscles. For example, the stimulation of the vertical recti generates elevation and small incycloduction, or depression and small excycloduction. Conversely, the stimulation of the oblique muscles produces small elevation and excycloduction or small depression and incycloduction. These combinations suggest the possibility of distinct nerve stimulations for the activation of these muscles as diagnostic elements of their functions. For example, elevation causes the activation (contraction) of the superior rectus and of the inferior oblique (and the relaxation of their antagonists), whereas the stimulation for the incycloduction of that eye through a reflex pathway by tilting the head toward the ipsilateral shoulder causes the activation (contraction) of the superior rectus and of the superior oblique (and the relaxation of their antagonists). Elevation in abduction, with tilting of the head toward the ipsilateral shoulder (i.e., an eye movement made in a direction that corresponds to the horizontal) would therefore lead to greater stimulation of the contraction of the superior rectus specifically. Thus, the diagnostic ocular rotations corresponding to the maximum innervation for the other underactive muscles in cases of vertical deviations would be in horizontal directions (on head tilting) (Figures 3 and 4).

A1) Application of the concepts of active forces

It is precisely the possibility of potential activations of the cyclovertical muscles in different positions of the gaze that generated Parks' proposal[42] for the differential diagnosis of which of them could be used to demonstrate the predominant muscle underaction in cyclovertical strabismus. It is unequivocally an elegant and simple test based on three steps. The first step corresponds to the simple determination of the type of deviation (L/R or R/L) to begin the reasoning based on only four (potentially underactive) muscles among the eight cyclovertical muscles under study. For example, in a R/L deviation, EOMs considered as potentially underactive are the right inferior rectus (RIR), the right superior oblique (RSO), the left inferior oblique (LIO), and the left superior rectus (LSR). The second step is measuring the deviation in the lateroversions (to the right and to the left). If, for example, the deviation is greater in levoversion, the predominant underaction must be ascribed to the RSO or to the LSR. Then, there is the third and last step, with head tilting to the right (greater stimulation of the RSO) or to the left (greater stimulation of the LSR), known as *Bielschowsky's maneuver*[43]*.* However, it should be noted that Bielschowsky proposed the maneuver specifically and only for the diagnosis of a superior oblique muscle palsy.

Notwithstanding, there are conceptual flaws in (a) studying diagnostic positions of cyclovertical muscles by testing their functions through *ocular movements in the horizontal plane* (second step of Parks' maneuver) and not through their classical diagnostic positions, wherein the innervation supplied to them is substantially greater; (b) considering the function of these muscles in Bielschowsky's maneuver but with fixation oriented by *straight ahead gaze* (third step of Parks' maneuver), i.e., with "attenuated" stimulations rather than "accentuated" stimulations, in both cases (second and third steps of the maneuver); and finally, (c) not specifying *which eye should be fixating* in each condition. Thus, there is no logical basis for this widespread test (Parks' test). However, its greatest flaw will discussed below ("diagnostic pairings").

Indeed, it was by the disciplined and constant use of this test in the diagnosis of cyclovertical deviations that illustrative examples of the gross errors in its conception and application were found[44-46], which was confirmed by other authors[47-48]. But to show the shortcomings of Parks' test in the diagnosis of vertical deviations, the examples presented by his own group to the American Academy of Ophthalmology[49] (Figure 2) may suffice, which, by the way, support the alternative proposal suggested by me (see Figures 3 and 4).

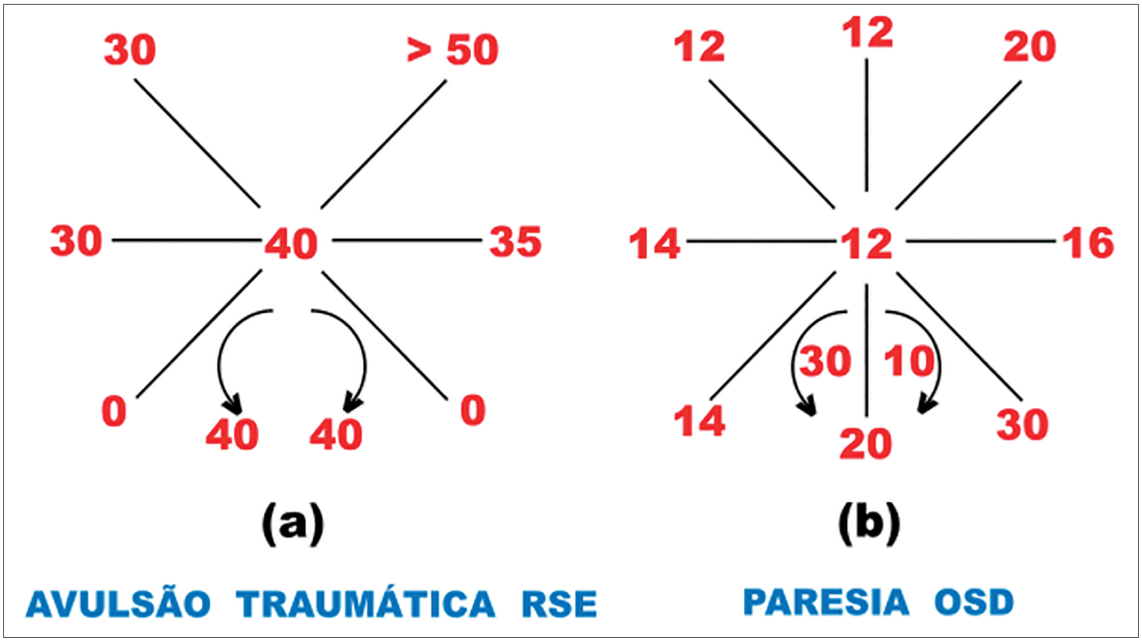


Figure 2. Parks method measurements obtained by Parks et al.[49] in a case of traumatic avulsion of the left superior rectus muscle (a) and paresis of the right superior oblique muscle (b). It should be noted in (a) that although the superior rectus has been detached from the sclera, there are no significant differences of deviations in dextroversion (30^{Δ}) and levoversion (35^{Δ}) or in deviations with the head tilted to the right (40^{Δ}) and to the left (40^{Δ}); however, in upper levoversion (> 50^{Δ}) and lower dextroversion (0^{Δ}) diagonal, the difference is enormous and greater than in the other diagonal 30 - 0 = 30^{Δ}. In (b), there is no significant difference between the measurements of vertical deviations in dextroversion (14^{Δ}) and levoversion (16^{Δ}), rendering Parks' test inconclusive between RSO and LSR. It should be noted, however, that the difference between measurements in lower levoversion (30^{Δ}) and upper dextroversion (12^{Δ}), i.e., 18^{Δ}, is significantly greater than that found in the other diagonal (20°- 14 = 6^{Δ}).

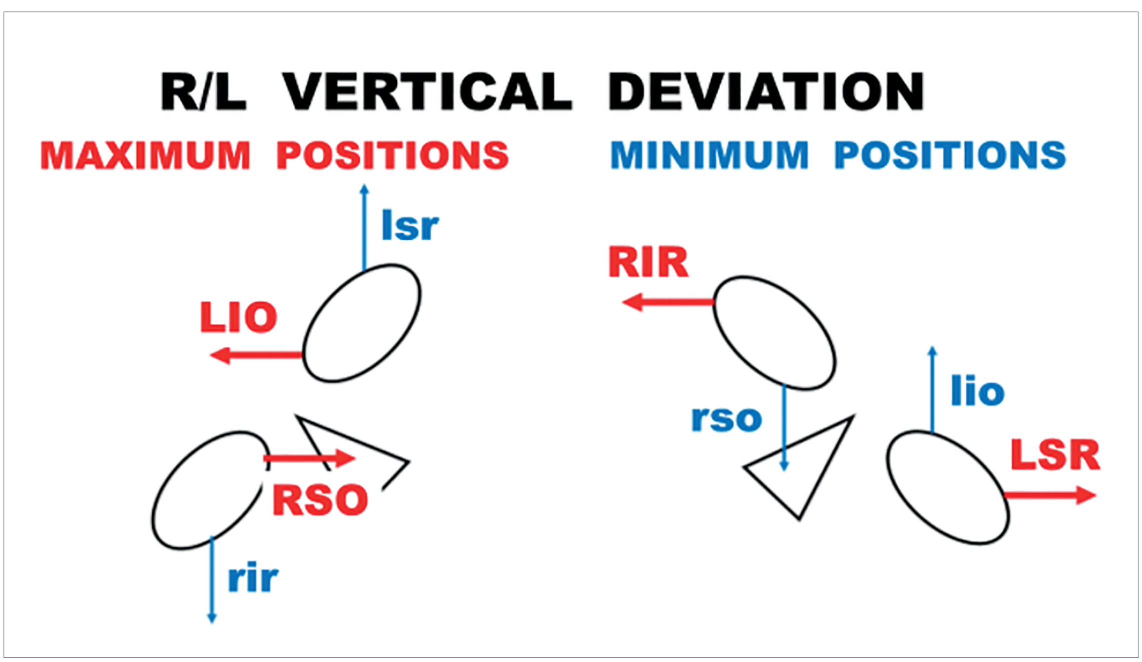


Figure 3. Diagnostic positions in which the highest stimulation for contraction of a cyclovertical muscle (in red) occurs and the respective position in which the stimulation is smaller (in blue) in a R/L vertical deviation. Notably, the direction of greatest stimulation for each possibly underactive muscle in this R/L vertical deviation (LSR, LIO, RIR, and RSO) corresponds to that of their respective classical diagnostic positions (upper, lower, lateroversion); but with the specific inclination of the head so that the stimulus becomes even greater; in such a condition, the respective direction of study will be *horizontal* (red arrow). For example, for the LSR, upper levoversion, with the head tilted to the left (fixating with the left eye), will correspond to a *horizontal* movement to the *left*.

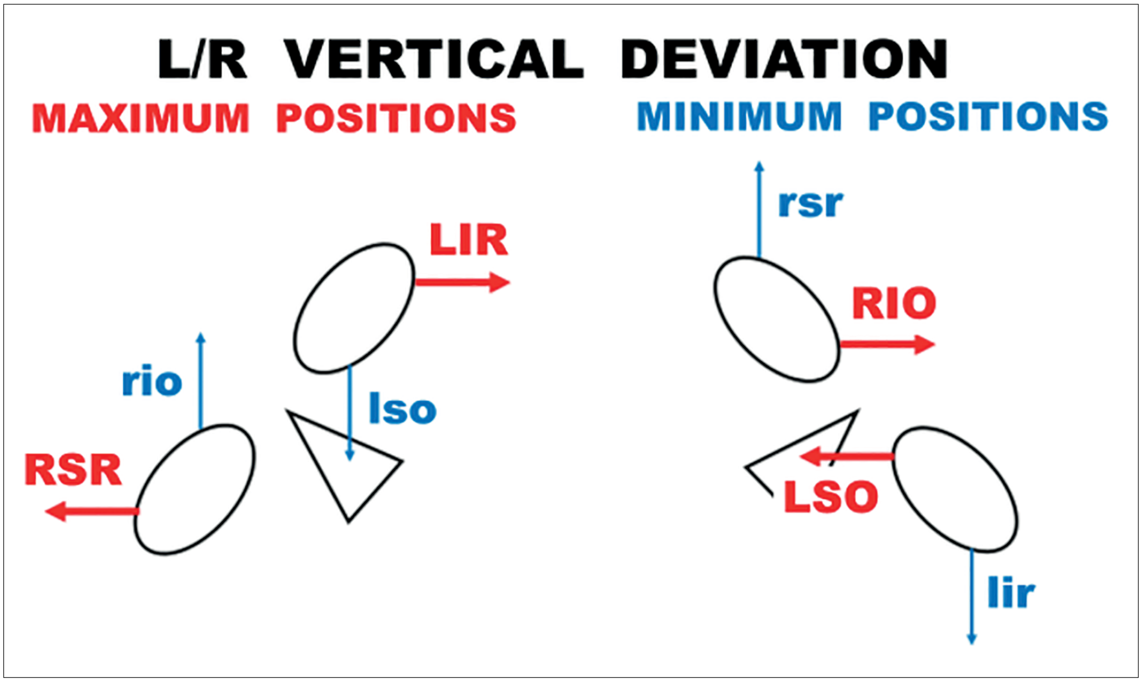


Figure 4. Diagnostic positions in which the highest stimulation for contraction of a cyclovertical muscle (in red) occurs and the respective position in which the stimulation is smaller (in blue) in a L/R vertical deviation. Notably, the direction of greatest stimulation for each possibly underactive muscle in this L/R vertical deviation (RSR, RIO, LIR, and LSO) corresponds to that of their respective classical diagnostic positions (upper, lower, lateroversion); but with the specific inclination of the head so that the stimulus becomes even greater; in such a condition, the respective direction of study will be *horizontal* (red arrow). For example, for the RIO, upper levoversion with the head tilted to the left (fixating with the right eye) will correspond to a *horizontal* movement to the *left*.

A2) Diagnostic pairings

Two practical conclusions emerge from Hering's and Sherrington' laws, which have been completely accepted, for the diagnosis of an underactive muscle in strabismus (due to paresis or action restricted by opposite forces), which are as follows:

a) Deviation is more marked when the fixing eye contains the (most) underactive muscle.
b) Deviation is greater in the direction of the action of the (most) underactive muscle.

Therefore, in paresis of the right lateral rectus, deviation is more marked with right-eye fixing and in right gaze (abduction of the right eye).

In fact, these two "golden" rules for the diagnosis of strabismus do not stipulate that the deviation only exists in these conditions but, rather, that it is *most* pronounced in them; nor do they state that only one muscle is underactive. It follows that, in this specific case, the deviation should be less marked under opposite conditions, i.e., two other complementary "golden" rules are valid:

c) The deviation is less marked when the fixing eye does not contain the (most) underactive muscle.
d) The deviation is less marked in the direction opposite to that of the action of the (most) underactive muscle.

This means that in paresis of the right lateral rectus, the deviation is less marked with left eye fixing and in left gaze. However, this condition is precisely that wherein a deviation caused by the paresis of the left lateral rectus would be more pronounced. On the other side, in paresis of the left lateral rectus, the deviation is *less* marked (in right gaze and with right-eye fixing) precisely in the conditions wherein paresis of the *right* lateral rectus would cause *more* pronounced deviation*.* In other words, these two muscles (RLR and LLR) form a ***diagnostic pair*** wherein the conditions under which they should be maximally and minimally stimulated are *antagonistic.*

In fact, in an *esodeviation*, the underactive muscle(s) (due to paresis or restriction) should be RLR, LLR, or both. Thus, only *two* diagnostic conditions need to be studied, i.e., that of the RLR (deviation in right gaze, with the right-eye fixing) and that of the LLR (deviation in left gaze, with the left eye fixing). If there is a clinically significant difference between them, the greater deviation indicates the muscle whose action is more deficient. If they are relatively similar, it cannot be concluded that one of the muscle's underaction predominates the other.

Similarly, for an *exodeviation*, the underactive muscle(s) (due to paresis or restriction) should be RMR, LMR, or both. Thus, only *two* diagnostic conditions need to be studied, i.e., that of the RMR (deviation in left gaze with the right-eye fixing) and that of the LMR (deviation in right gaze with the left eye fixing). If there is a clinically significant difference between them, the greater deviation indicates the muscle whose action is more deficient. If they are relatively similar, it cannot be concluded that one of the muscle's undercation predominates the other.

An identical procedure can be performed with regard to vertical deviations, wherein the action of the vertical recti predominates. In a R/L deviation, the test conditions would be those of RIR (deviation in downward gaze with the right-eye fixing) and those of LSR (deviation in upward gaze with the left eye fixing). In a L/R deviation, the test conditions would be those of LIR (deviation in downward gaze with the left eye fixing) and those of RSR (deviation in upward gaze with the right-eye fixing).

But what about the oblique muscles? The best condition to study them is through ocular torsions, which are difficult to provoke (they are not voluntarily engaged and their magnitudes are small through reflex pathways), difficult to observe (rotation around the longitudinal ocular axis), and to measure (prisms do not apply to them). Therefore, the procedure in clinical practice includes assessing the function of the oblique muscles using their horizontal components (e.g., the variation of the horizontal deviation in the sagittal plane; the so-called *horizontal* variations in "A" or "V") or, primarily, their vertical components. Vertical deviations caused by the oblique muscles are not very pronounced but are greater in adduction than in abduction (wherein the predominant action of the vertical recti is even greater), and this is therefore an option to study these muscles. Thus, a vertical deviation caused by the underaction of the the RSO, i.e., a R/L deviation, is *greater* in downward gaze and to the *left* with the right eye fixing, and is *less* marked in upward gaze and to the *right* with the left eye fixing, a condition that is related to the greatest deviation originated by an underaction of the LIO. These two antagonistic conditions correspond, therefore, to the diagnostic pair RSO-LIO in the R/L deviation. In this deviation the pair of vertical recti (RIR and LSR), is also involved, whereas in the L/R deviation two diagnostic pairs (pair of obliques and pair of vertical recti) should be assessed (Table 1).

Table 1. Positions of the greatest and of the smallest stimulation of the vertical recti and oblique muscles in vertical deviations.

DEVIATION	MUSCLE	CONDITION OF STIMULATION	
		GREATEST	SMALLEST
R/L	RIR	Downward/right, RE fixing	Upward/left, LE fixing
R/L	LSR	Upward/left, LE fixing	Downward/right, RE fixing
R/L	RSO	Downward/left, RE fixing	Upward/right, LE fixing
R/L	LIO	Upward/right, LE fixing	Downward/left, RE fixing
L/R	LIR	Downward/left, LE fixing	Upward/right, RE fixing
L/R	RSR	Upward/right, RE fixing	Downward/left, LE fixing
L/R	LSO	Downward/right, LE fixing	Upward/left, RE fixing
L/R	RIO	Upward/left, RE fixing	Downward/right, LE fixing

However, the maximum stimulation can be even further accentuated if the labyrinthine stimulation of ocular torsion is triggered by adequate head tilting. Indeed, on right head tilting, the incycloductors of the right eye (RSO and RSR) and the excycloductors of the left eye (LIO and LIR) are stimulated. On left head tilting, the incycloductors of the left eye (LSO and LSR) and the excycloductors of the right eye (RIO and RIR) are stimulated. The combination of the stimulation of a muscle through ocular rotation to its classical "diagnostic position" with an adequate head tilt resulted in the proposal of studying the *diagnostic positions with the head tilted*[50-54] (Figures 3 and 4).

As the author of this concept of diagnostic methods in strabismus, especially that of diagnostic pairs, I was distinguished with an invitation to present them at the I.S.A.-C.L.A.D.E. conference in the joint congress of these two entities[55].

A3) Rotations in the opposite direction relative to the original

Another simple and direct application of the concept that all muscles can "produce" all (any) actions is the use of forces of the ***medial rectus*** muscle (i.e., a muscle ascribed as purely concerned to adduction) to produce ***abduction.*** In fact, assuming a complete loss of action of the lateral rectus muscle due to its anatomic absence or to an innervational palsy, the principle of recovering its action is to "anchor" the eye to the orbit in a position of abduction (the desired position to which the eye may reach) through an extendable device like a spring or an elastic. Through the innervational stimulation of the medial rectus muscle the eye can be brought to the primary position by a centripetal rotation (and can even surpass it in a greater adduction). This movement of centripetal adduction leads to the accumulation of potential energy (originated from the *medial* rectus muscle) in the expandable elastic device, and that energy can be recovered later when the medial rectus muscle relaxes, promoting abduction (centripetal and/or centrifugal depending on the point from where it starts). The theoretical foundation of this idea was developed in a sequence of publications[56-62], and its application was explored in studies conducted by me in Ribeirão Preto[63, 64] and by Collins et al. in San Francisco[65-67], with the difference being solely the orientation of the traction. In fact, a "direct" traction of the device was preferred at the Smith-Kettlewell Eye Research Institute (S.K.E.R.I.) to produce abduction: the agent for pulling the eye was attached to the temporal sclera in front of the center of ocular rotation, and it was attached to the orbit behind this center of rotation, on the temporal side, a technique with which I started (four patients operated in 1982 and one patient operated in 1983 with the implantation of helicoidal steel springs)[63] (Figure 5). Subsequently, I used a "reverse" double traction: the agents for pulling the eye (silicone threads) were fixed to the sclera on the *medial* side in front of the center of ocular rotation, and to the orbit in *front* of the center of ocular rotation on the temporal side, by two fascicles (a superior fascicle and an inferior fascicle)[64].

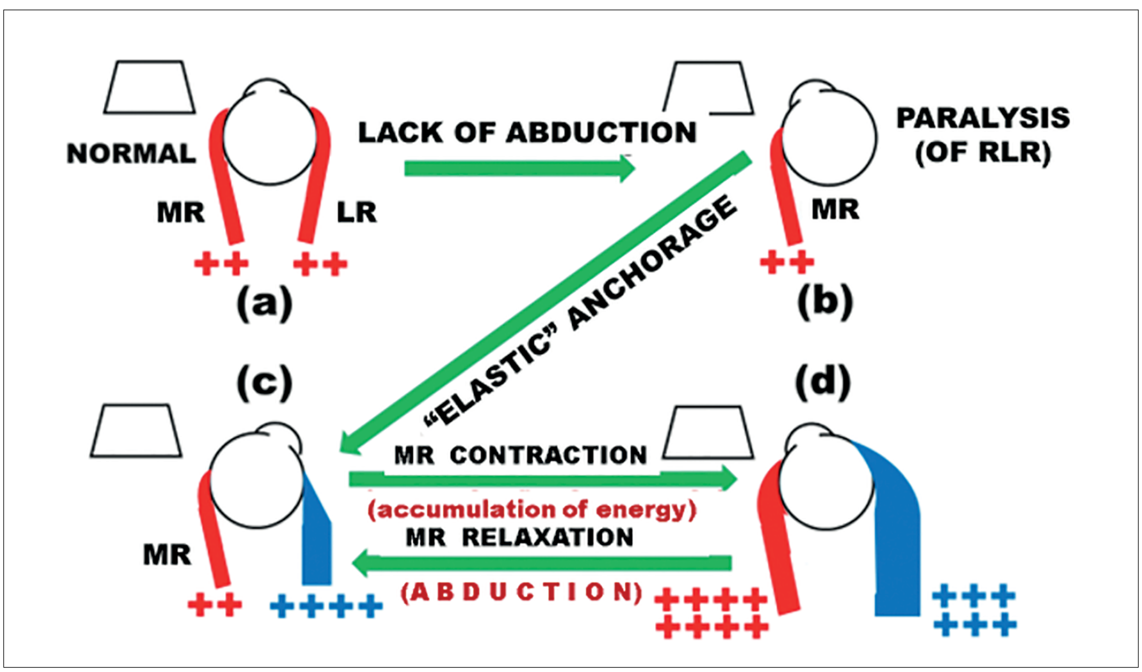


Figure 5. Demonstrative scheme of the method for restoring ocular rotation (in this case, of the RLR) by forces originating from its antagonist (RMR). (a): Normal state of RMR and RLR muscle tonicity (++ in red). (b): loss of RLR action, esotropia due to the predominance of RMR action. (c) anchoring of the eye in abduction by greater tension of passive forces (++++ in blue). (d) position after increased force by RMR contraction (+2 to +8), overcoming passive forces, which also increase by elastic artifact distension (+4 to +6). From this state (d), by *relaxation* of the RMR (from +8 to +2), the elastic "shortens" (from +6 to +4) and its (passive) forces are released, so that the initial state (c) is restored, with abduction. Note that the accumulated and now released passive forces (6 - 4 = 2) are consumed in the passive stretching of the medial rectus (+2), adding to its relaxation forces (+4) to explain the difference in their tensions (6 = 8 - 2).

It is interesting how in the excitement of rescuing ductions, the *monocular* results, which, by the way, were satisfactory and confirmed the principles, blinded us all for so long, to the extent that we did not pay attention to *another* basic principle. We (Scott, Miller, and Collins from the S.K.E.R.I, and myself) were invited to participate in a closed and very high-level meeting in San Francisco, focusing on the engineering of ocular movements. Because we were the only speakers in the section of "ocular prostheses," I felt obliged to present an in-depth theoretical insight into the topic to befit the attending scholars, and therefore, I talked

about *binocular* movements*,* or *versions*[68]. To my constraint, I realized, through *theory* and not through a simple *observation*, that while the adduction movement of the treated eye is hampered (by the elastic restriction), Hering's and Sherrington's laws apply to the normal eye, as intended by nature, and cause it a wider movement. Hence, there is no possibility of obtaining *conjugated* binocular movements, unless (but this is unthinkable) one creates a "conjugated" defect in the "good" eye to hinder its *adduction*, i.e., paralyzing the corresponding adduction of the normal eye and "correcting" it by using an elastic device. My consolation was that I realized this before the researchers of S.K.E.R.I., who, at the start of their presentation (which was scheduled following mine), were already deeming it outdated[69]. This line of research has then been shut, but the idea that a muscle could provide a rotation in the direction opposite to that which is normally expected remained.

B) Passive forces

With the exception of the examination of a possible rotational restriction of the ocular movements by the "test of passive ductions" to confirm it or rule it out, passive forces were not considered to have an important role in oculomotor balance. But by the study with which the accumulated forces during such passive ductions could produce ocular movements in the absence of muscle tones (active forces) --- for example under general anesthesia during surgery to correct strabismus, i.e., the study of centripetal rotations or (passive) spring-back rotations[70] --- allowed the development of a new method to study the forces involved in oculomotor balance. Therefore, analysis of the *centripetal* movement was now being performed instead of (or complementary to) the simple assessment of "resistance" to *centrifugal* ocular rotation. While, previously, surgery was only seen as a therapeutic procedure, from that time it also gained a *diagnostic* value due to the improving knowledge of the problems that caused visual axis deviation and planning of their correction. It is very likely that the term "passive forces" was used for the first time in the rationale for this method[71].

The *point of passive balance*, or the measure of the deviation under general anesthesia, is determined by the mean of the final positions of the centripetal movements in opposite directions (for example, from symmetrical points of passive abduction and passive adduction). Thus, for a deviation measuring +20° (in the waking state), a distinction can be made between a "purely tonic" deviation (when, under anesthesia, the measure of the deviation is 0°), a "mechanical" deviation (when, under anesthesia, the deviation remains identical, i.e., +20°), and a "mixed" deviation (when, under anesthesia, the deviation is between 0° and +20°). In addition, the observation of the amplitude and/or the velocity of the spring-back "centripetal" movements (which may vary, but, usually, are directly proportional to each other), could allow collecting information regarding the contraction capacity of the muscles (related to their elasticity). For instance, if the point of passive balance was 0° but resulted from centripetal rotations ending in +10° (from the centrifugal adduction) and −10° (from the centrifugal abduction), the condition should be deemed as *different* from another measure with the *same* point of passive balance (0°), but obtained from the mean of centripetal rotations ending in +20° and −20°. In the former case (greater centripetal rotations), this would mean more "elastic" or tense muscles, which would therefore respond *more* to procedures that debilitate or strengthen their actions and, therefore, bring about *greater* surgical results than those obtained with *identical* procedures in the latter case (less elastic or looser muscles). The unquestionable *prognostic* improvement provided by the results of these surgical (but diagnostic) procedures altered the conduct of performing the surgery based solely on a previously established plan and going forward with it, without any modifications. In summary, peroperative data based on the acting "passive" forces guided not only the selection of the muscles that should undergo intervention but also the extent of these interventions.

B1) Muscle elasticity as an explanation of surgical results

An interesting surgery introduced at that time was the Faden operation[72] (German for "thread operation"), wherein the eye was fixed to the sclera, far from its anatomic insertion, without actually detaching it. The surgery was theoretically equivalent to a recession of the insertion (a "new" insertion, relative to the original one) but with a "magnitude" equal to that of a resection of the same value (muscle tension should not be altered with the procedure). The aim was to reduce the arc of *functional* contact by correcting, for example, an esodeviation in near fixation, without changing the oculomotor balance in far fixation. An unexpected problem was that the modification of deviation in far fixation *also* occurred. Moreover, the values of correction of deviations were, apparently, not related to the "magnitude" of the operation (that is, the distance between the original insertion and the new muscle-scleral suture).

In 1981, I was invited to discuss a presentation of De Decker (probably the most renowned about the subject and with its greatest casuistry) at the Wenner-Gren Center Symposium (Stockholm)[73], and I suggested that the explanation for the apparent incongruities should probably lay in the manner the surgery was performed. In fact, the suture was not made with the eye in the primary position, which in effect would not change muscle tension, but rather by pulling it to bring forward the region where the muscle would be sutured to the sclera, to ease the procedure. Therefore, the muscle was *distended* in a manner that the site of its insertion into the sclera corresponded to a recession that was effectively *greater* than it appeared. This difference basically depended both on the extent to which the eye was rotated and on the elasticity of muscle (Figure 6)[74].

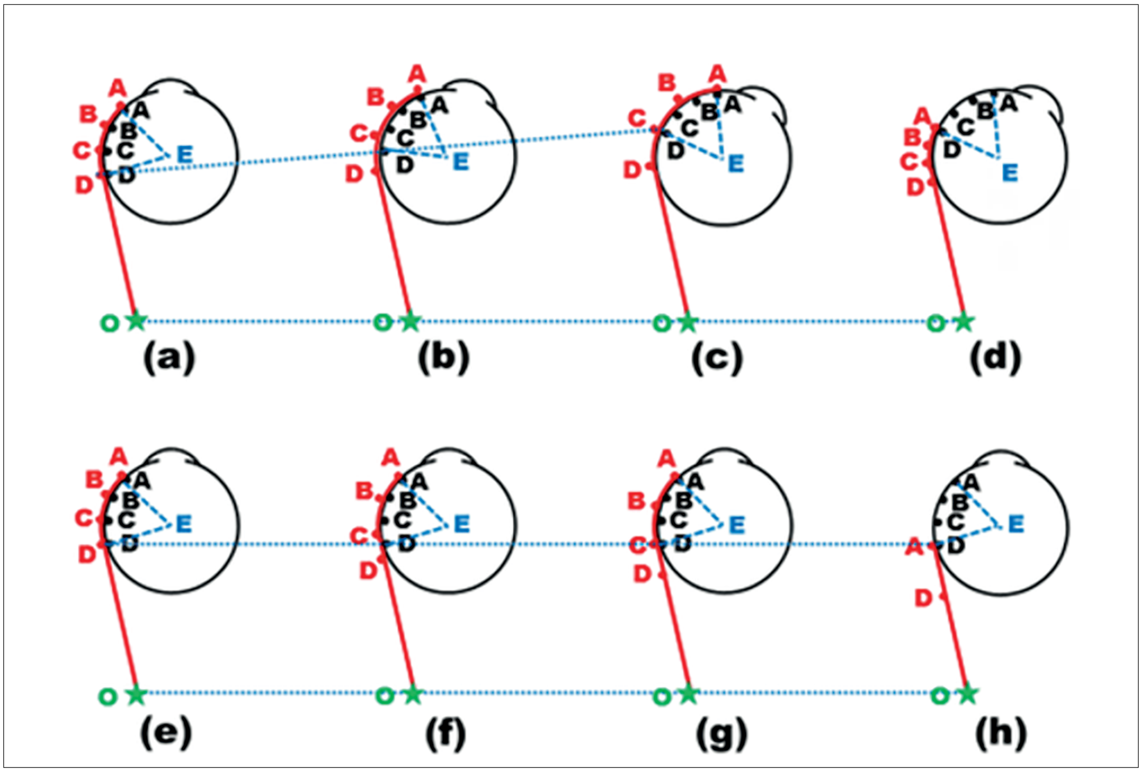


Figure 6. Explanatory scheme of the unequal effect of apparently identical faden operations (a), (b), (c) and of a muscular insertion recession (d) (in this case, identical to that of the faden operations). In the primary position of gaze (a), the muscle points of contact (A, B, C, and D, shown in red) and the sclera (the same, but in black) coincide. If the eye is passively moved in the opposite direction to that of the muscle action (b, c), the muscle (red line) is assumed to proportionally stretch to its length (between the scleral insertion point of the muscle, A, and its origin, O), being smaller (b) or larger (c) depending on the rotation generated. Notably, when the muscle is distended, its points (in red) and the corresponding points in the sclera (in black) that previously coincided fail to do so at distances proportional to the muscular stretching. As the magnitude of the faden operation is determined by the size of the scleral arc (or its corresponding chord), for the same values of the arc (AED) or the chord (AD), the point of the muscle sutured to the D point of the sclera will be D (in "a"), between C and D (in "b"), C (in "c"), and A (in "d"). Thus, in the primary position of gaze (respectively "e," "f," "g," and "h"), the results of these apparently identical operations (by the same AED scleral arc) will be different depending on how much the muscle was stretched. Notably, although the distance D (from sclera) to O is the same after the procedures ("e," "f," "g," and "h"), the distance from the point D from the muscle to the origin (O) decreases under these conditions.

B2) Influence of other periocular structures on eye rotations

The influence of the structures surrounding the EOMs and between them (fasciae and intermuscular membranes) on muscle actions was an additional object of analysis. The theoretical calculations with regard to each of them (corresponding to the eye rotation from the primary position of gaze to a specific direction in space)[34-39] implied that in the desired ocular movement, the muscle could slide freely over the sclera, always occupying the arc of a *maximum circle* (the smallest distance between two points on the surface of a sphere). However, this displacement is limited by the fasciae and intermuscular membranes to a greater or lesser degree. The concept that is currently discussed as *muscle pulleys* was already foreseen back in 1978, when "the effect of structures of restriction on the direction of the force of application of a muscle" was shown pictorially (Figure 8, p.15)[7]. The conclusion was that "the sectioning of intermuscular membranes would allow the muscle to slide more freely over the eyeball during its rotations, thereby modifying the distribution of forces between the distinct planes" (Figure 7).

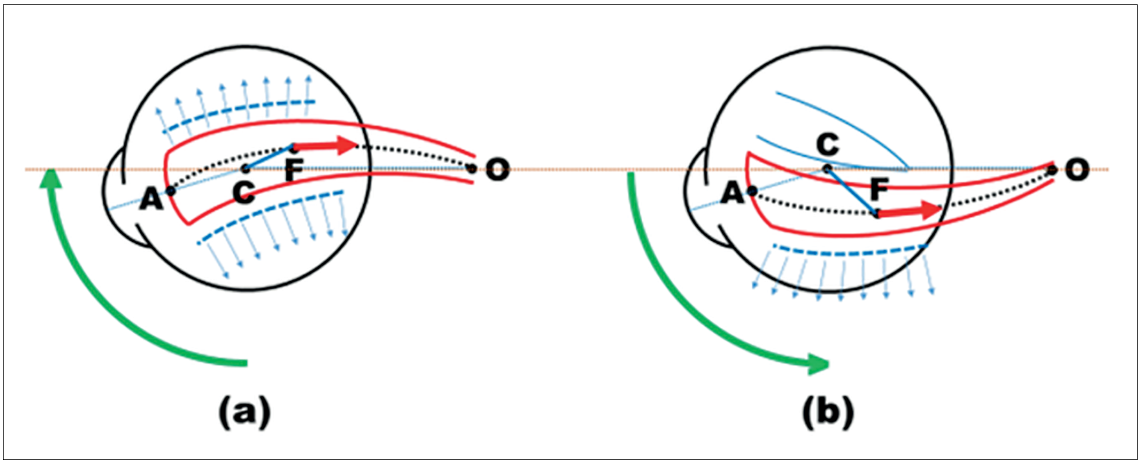


Figure 7. Schematic representation of the action of a horizontal muscle before (a) and after (b) an intermuscular membrane section (above the horizontal plane). From an antagonist of a downward ocular rotation (as an elevator because the center of ocular rotation is considered to be below the point of force application), the muscle becomes synergist. Notably, in the auxiliary horizontal line, C and O are assumed to be in the same plane before and after the membrane section.

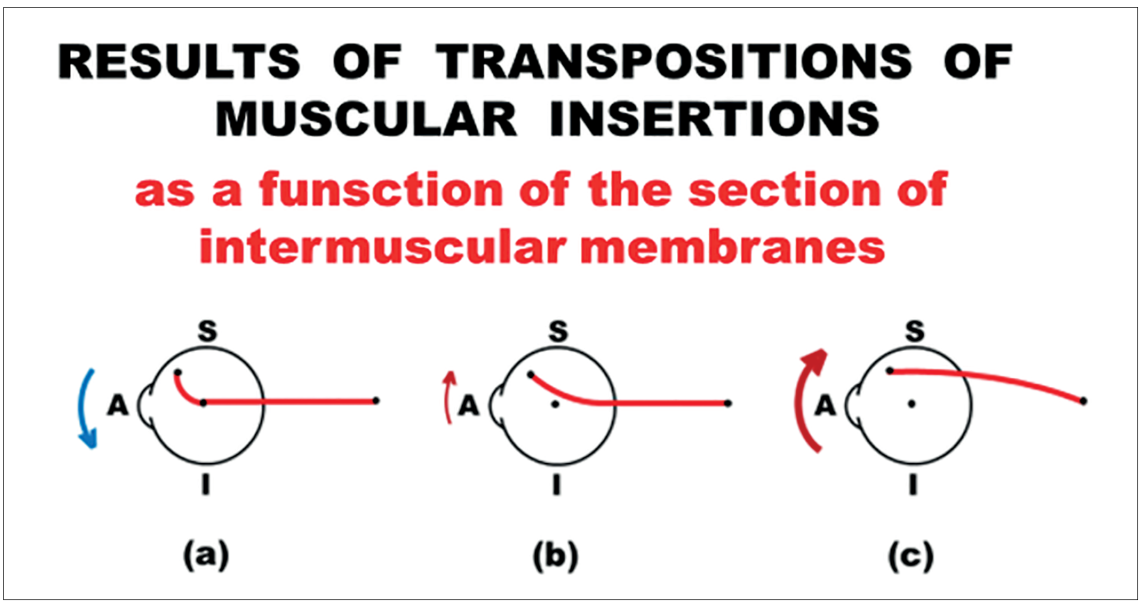


Figure 8. Schematic representation of the change in action of a horizontal muscle transposed over the horizontal plane according to whether the intermuscular membranes are intact (a) or sectioned into a small (b) or large (c) extension. In the first case (a), although transposed "above" the equator, the muscle is a depressor. In the wide section of the intermuscular membranes, longitudinal to the muscle, its path becomes a "great circle," with an increase in the angle of force application (c), and the muscle acts as an elevator. At the intermediate level, this force application angle is smaller and the muscle action of elevation is also smaller (b).

This notion of the point of application of force on the eye was reintroduced in a more generic presentation on ocular kinetics (in another Wenner-Gren International Symposium in Stockholm, in 1987). Depending on whether the sectioning of the intermuscular membranes is performed (or not) and how it is performed, the direction of a muscle's action may be inverted[76]. At the Master Conference of the Congress of the 25th Anniversary of the Spanish Strabismological Association (Madrid, 1996), although this made up a minor part of the presentation, new graphical elements were added to show how the status of integrity of these membranes that act as "pulleys" influences the positioning of a muscle on the eye, both by altering its action in the various gaze directions and by having an effect on its potential transpositions[77,78] (Figure 8).

However, the presentation of 1987 in Stockholm[76] also included discussions about concepts, such as the "arc of rotation" which had also been foreseen[61] to replace the concept of "arc of contact," that still prevailed; as well as the influence that ocular translations, even if small, have on ocular rotations, namely by limiting them.

C) Clinical applications

A summary of the mechanisms of action of various surgical procedures for the correction of strabismus was presented in a lecture at the XII CLADE Congress (Buenos Aires, 1996). The different methods and their fundamentals were assessed with respect to the following:

A) Changes in the plane of muscular action
1) Changes in the relative position of the muscle and eye;
2) Changes in muscle length;
3) Rigid spacer;
4) Myoscleropexias (faden operations);
5) Association of resection and hang loose sutures;
6) New combinations.

B) Changes of the plane of muscular action
1) Transpositions;
2) Relative transpositions;
3) Oblique insertions.

C) Rotational moment changes
1) Changes in contractile force;
2) Changes in the moment arm;
3) Changes in the angle of force application.

D) Effects of non-muscular structures
1) Influence of connections between muscle and eye (foot-plates);
2) Influence of connections between muscles (intermuscular membranes);
3) Influence of connections between the orbit and eye or its muscles;
4) Restraining devices.

The difference between “changes *in* the plane of muscular action” and “changes *of* the plane of muscular action” can be determined by the amount of the insertion recession. In fact, a simple recession, *with no change of the plane of muscular action* (Figure 9), leads to possible changes in muscular action[75].

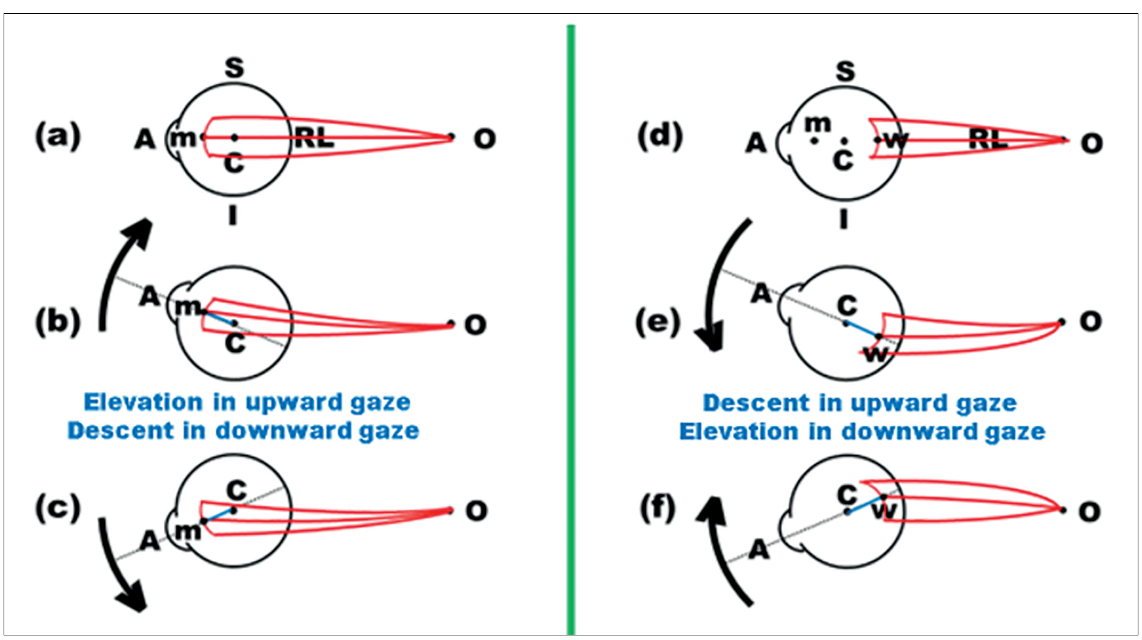


Figure 9. Change in the action of a horizontal rectus muscle (LR) by a large recession of its insertion (from *m* to *w*) (intermuscular membranes are supposed to be sectioned). With the normal insertion (a), there is a small vertical action of this muscle, but ocular elevation in sursumduction (b) and lowering action in deorsumduction (c); after a recession in which the reinsertion is made behind the center of ocular rotation (C), *i.e.*, in *w* (d), there is a small vertical action of this muscle, but of depressing the eye in sursumduction (e) and of elevation in deorsumduction (f). That is, the muscle goes from being a synergist of the vertical rotations to becoming an antagonist.

Among several other methods (some of which are original), is noteworthy the bifid insertion of a muscle (done symmetrically with respect to its original plan of action) in order to weaken the muscular action by reducing the moment arm and the angle of application of the force[79] (Figure 10).

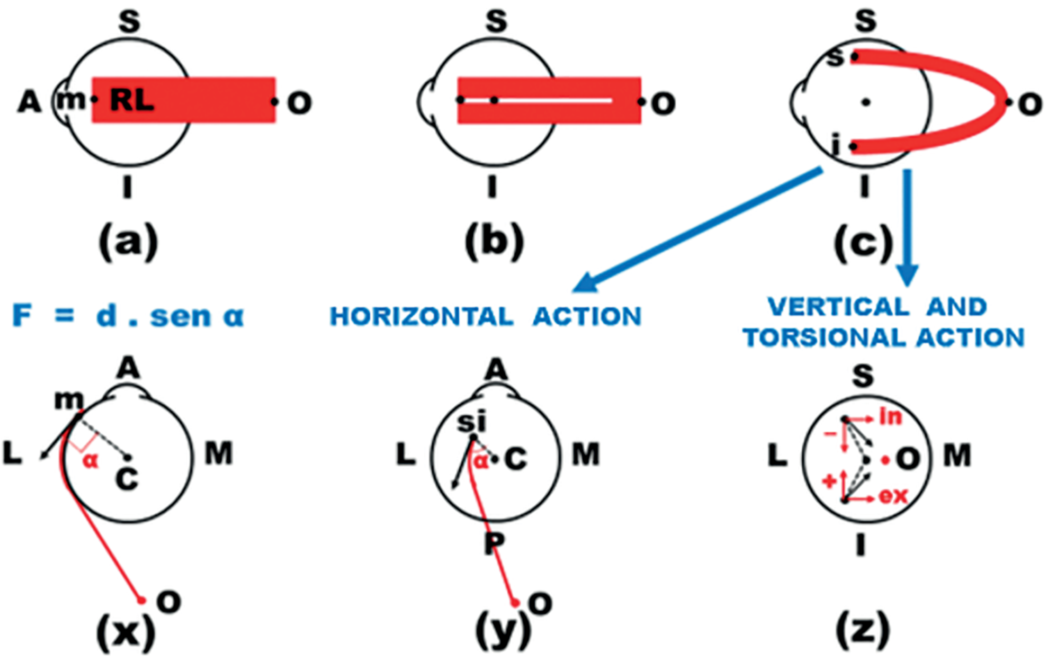


Figure 10. Bifid insertion surgery of a muscle to reduce the angle of application of force (α) and its moment arm (d) so that the resulting force applied to the eye (F = d.sin α) becomes smaller or even zero (when d = 0 or α = 0). Schematic representations of the left eye in lateral view, with the muscle (LR) before the intervention (a), with longitudinal separation of the muscle (LR) into two symmetrical portions (b), and after reinsertion of the upper (s) and lower (i) flaps in symmetrical positions above and below the horizontal plane (c). Schematic representations of the position of the muscle (red trace) relative to the center of ocular rotation (C) in the horizontal plane before intervention (x) and after (y); in the frontal plane (seen from behind) after the intervention (z); the torsional actions of incycloduction (in) and excycloduction (ex) cancel each other out, similarly to what happens with sursumduction (+) and deosursumduction (-).

A proposition to increase the arm with which the force is applied to the eye is also considered as a possibility of changing the moment of rotation; the possible advantages and disadvantages of this technique are discussed. In this paper, the use of *"atropine in dilutions of 0.01%–0.05% to produce cycloparesis and thus increase the AC/A ratio"* is also suggested in order to change the innervation command (to change the contractile force of the muscle) (Bicas et al., 1978).[80] Indeed, this atropine dilution was probably used for the first time in ophthalmology clinics at that time, 40 years ago. Nowadays, it is commonly used, but for another purpose, that of the prevention of myopia progression.

Another analytical review presented at the Smith-Kettlewell Oculomotor Symposium, held during the 1996 CLADE Congress, addressed the concepts of "*elasticity, stiffness, and other terms*" and their corresponding clinical applications.[81]

D) Dissipative Forces

Ocular rotations are determined by increases or decreases in the tonicities of the extraocular muscles (during its *contractions* or *relaxations*, respectively); these rotations are always mediated by stimuli from the central nervous system. The forces from which they originate, the so-called *active forces* (A), would continue indefinitely if there were no other forces, the so-called *passive forces* (P) or *reaction forces*, to oppose them and determine the end of movement. Muscles and other elastic structures, which elongate during a centrifugal rotation, oppose the continuity of motion and determine its interruption, accumulating the kinetic energy of motion, without requiring a new innervational stimuli to be triggered to stop the movement. The established balance then continues as long as the command for contraction of the stimulated muscle(s) persists. During relaxation of these muscles, the passive forces produce centripetal ocular rotation (spring-back rotation), which would continue as a centrifugal rotation until, again, the elastic structures providing resistance to the continuity of rotation (promoting the end of the centrifugal rotation) determine its stasis and the beginning of a new cycle, maintaining an oscillating (pendulous), harmonic movement.

However, this is not how things happen; for abduction of a certain value, it is necessary to stimulate the lateral rectus by a certain magnitude such that the rotation is limited by passive forces. When relaxation occurs, the centrifugal movement "spontaneously" ends after the centripetal rotation, without the centrifugal rotation starting and with no new innervation stimulus being triggered for the stasis. The explanation is simple; in addition to the *active* and *passive* forces, the oculomotor system has *dissipative* forces (D), which consume kinetic energy and transform it into heat (during friction between the moving surfaces and by distension of elastic tissues) and inelastic deformations. The equation is even simple: the energy accumulated by the passive forces is the difference between that of the active and dissipated forces (P = A − D), while the energy of the spring-back rotation equals the dissipated energy (P = D). Thus, the magnitude of the dissipative forces is usually about of the original force imparted by the muscle triggered by the innervation stimulus (A = 2 D).

In addition to the *quantitative* aspect, the *qualitative* importance of the dissipative forces (D) is obvious; they "clear" the oculomotor system of the forces generated by a given innervation stimulus (A), allowing a new one to occur, without the need for any other renewing *order*. These forces are "destructive" and hence "renovating." The idea that they can be harnessed to "destroy" (unwanted) rotations, such as those of nystagmus, is evident. This was hypothesized,[82] and experiments that could substantiate this hypothesis were initiated. The proposition on which André A. H. Jorge's doctoral thesis was based, which has subsequently been expanded[82,83] and of which I had the honor of being the advisor, was justly awarded the annual Oftam–CBE Prize in 2001. New studies were published[84-87] (two other doctoral theses I advised)[85,87] and later awarded the Waldemar and Rubens Belfort Mattos Prize in 2010 for best paper published in the Arquivos Brasileiros de Oftalmologia (Brazilian Archives of Ophthalmology) in the previous year[86].

E) Forces of Magnetic Fields

In short, one can consider strabismus as being produced by two elementary causes: mechanical restraints to ocular rotation (*e.g.*, those produced by orbital floor fracture and Brown's syndrome, among others) and functional deficiencies in innervation (*e.g.*, paresis and oculomotor paralysis) and/or in muscular reaction (*e.g.*, myopathies, muscle avulsions). In the former case, it is necessary to try to eliminate the restrictive forces. In the latter case (innervation or muscular response), the oculomotor balance may be eventually restored by weakening opposed muscular actions (insertion recessions, botulinum toxin applications, myotomies, or myectomies) or by *increasing* pre-existing rotational forces (muscle shortening, scleral reinsertion of detached muscles, greater convergence by stimulating accommodation). The principles of muscular transpositions to "restore" a lost rotation (for example, Hummelschein's surgery[88] or its variants) cannot withstand arguments that the response sought is only mechanical and "passive" (which, then, could be achieved by traditional procedures such as shortening the paralytic muscle), and have to admit that there are no re-learning commands (such as for abduction by the vertical recti muscles, which are synergists of *adduction*) so that greater oculomotor imbalances would have to be produced (e.g., unwanted vertical deviations in ocular rotations in the horizontal plane)[89,90].

Procedures by which *new* forces might be introduced into the oculomotor system (*e.g.*, electrical stimulation of paretic or paralytic muscles with signals picked up by rotations of the "normal" eye) have already been considered; however, these procedures present enormous technical difficulties, with possible tissue damage caused by the electric stimulation of the affected muscle, and do not present practical solutions that would encourage further studies. A theoretically possible alternative would be to make use of sufficiently strong "natural" forces (such as those of magnetic fields). This approach would have the advantage that such magnetic forces can be " regulated" by signals from the other eye or, simply, generated by small magnets. Speculations about this subject were initially made in 1985[62] and later retaken due to the theoretical failure of the application of elastic artifacts to replace lost ocular rotations.[68] New studies were conducted[91-97], and the first interventions in humans were reported[98,99], winning the Oftam–CBE prize in 1997[98]. Experimental improvements have been described[100-103], and two PhD theses on the subject have been completed under my advice[104,105].

Owing to my retirement from FMRPUSP and the conviction that my vitality to work would not allow me to reach the quantities and qualities of the academic production "required" by the evaluation criteria of post-graduate courses, I chose to conclude my formal teaching activities despite the remarkable and generous invitations from my colleagues to continue working at the Department, which immensely honored me. Therefore, I interrupted my research that, I still believe, have a promising solution not only for the stabilization of nystagmus but also for the stabilization of achieved results of strabismus surgeries. This hypothesis was debated at an international symposium at the Smith-Kettlewell Eye Research Institute (2012)[106], leading to a discussion on a possible wider clinical application[107]. (This article was quoted in a recent publication in the United Kingdom, which re-addresses the proposal for nystagmus correction, reproducing practically all of our original works and presenting yet another good result of the technique[108].)

ADD-ONS

Naturally, studies on "the strabismology I have lived" are not limited to those mentioned here. Academic life confronts us with multiple challenges, and several other studies have been shared, mainly through my advice in PhD theses and Master's degree dissertations, participations in books etc. Each of these experiences added to my learning. It is interesting to note that one subject that received particular attention (the objective register of eye movements by electro-oculography), --- which was my focus for our (academically) most important Thesis[109], which was applied to the first observations of spring-back rotations, when they began to be researched at the Smith-Kettlewell Institute of Visual Sciences (1974); which led to several presentations at international congresses[110-115] and publications[116,117]; which was the theme of the first PhD thesis under my advice[118]; and the subject of various courses and lectures --- have been later less valued, although did not lose its importance. Meanwhile, Master's degree dissertations and PhD theses have studied other subjects through different approaches (in addition to those previously mentioned) such as diagnostic methods during surgery[119-123], monocular and binocular sensorial studies[124,125], psychophysical studies[126,127], clinical methods for the diagnosis of strabismus[128-133], use of botulinum toxin[134,135], readjustable sutures[136], tissue adhesives[137], other surgical techniques[138-142], study of satellite cells[143]and, fortunately, some others, with distinguished colleagues. The diversity of these topics testifies to the evolution of the study of strabismus throughout the years.

Proverbially, "the practice of medicine is both science and art." Actually and specifically for strabismus practice, without losing focus on science, the art aspect possibly prevails. Indeed, for the same problems, different solutions have been proposed, each of them presenting reasonable amounts of good results, confusing the best explanations on why this might happen.

In fact, the conclusions of the Symposium on Strabismus of the XXI Brazilian Congress of Ophthalmology (Recife, 1981), which I had the honor of organizing and coordinating and for which I was awarded the Carmem Caldeira Prize by the Brazilian Center for Strabismus (CBE), was paradigmatic. I gave it the title "Knowledge and procedures on ocular motility and related functions considered indisputable," aiming to catalog what would be considered as "certainties" by 17 Brazilian strabismologists who were then recognized as the most experienced and successful in the field. They were asked by mail to list the statements that they considered to be absolutely correct and that could not be amended. The number of "certainties" then stated was large, exceeding five hundred (although many of them had the same essence). Subsequently, these "indisputable" statements were grouped into topics and were anonymized; the total list was redistributed to all the survey participants, who were asked to attribute values to each one, ranging from "ten" (when the statement was considered to be completely true) to "zero" (when it was considered to be absolutely wrong). It was expected that the authors would (naturally) assign "ten" to their own statements but also to some of those made by other colleagues, so that statements unanimously accepted with the maximum mark could represent the set of "certainties" of the CBE. Surprisingly, only *one* was unanimously accepted, and it basically stated that it was preferable to prevent amblyopia than to treat it, which was practically obvious. Evidently, the method for obtaining these truths is in itself vulnerable to criticism, because one might state, for example, that "the medial rectus is an adductor muscle," believing that this is an indisputable truth, while another might consider it questionable because this muscle *also* has a sagittal (vertical) action when the eye is outside the horizontal plane (and, therefore, may be attributed a sub-maximal evaluation to such statement). Nonetheless, this was a fair representation of the lack of consensus, if not in the concepts, at least in the language in which they are put.

In fact, the very definition of "strabismus" is not even consensual, giving rise to interesting controversies. For example, strabismus is often defined as the misalignment of the main visual axes ("foveal axes") relatively to the point of space over which they should intersect. This seems to be the most usual yet insufficient conception; the visual axes can intersect over the referential point in space but with inclinations between the respective *horizontal* (or sagittal) planes of the eye (*i.e.*, of vision), therefore without "horizontal and/or vertical deviations", but with *torsional* deviations. Furthermore, a good binocular positioning may be observed for one (or more) point(s) in space, but not for *any* (which imposes a spatial condition to the concept). Moreover, visual axes may be missing (blindness) and nonetheless the patient might be referred as "with strabismus" (because of deviations between *other* eye axes taken as a reference). In summary, "visual axis" is a convenient and probably necessary, but insufficient and sometimes inadequate, reference to define strabismus.

Perhaps it is a purism to claim that the practicality of strabismus diagnosis and therapy be subordinated to concepts. However, at the Keynote Lecture of the XXV Brazilian Congress of Ophthalmology[144] (Belo Horizonte, 2009), --- a privilege granted to the President of Brazilian Council of Ophthalmology in the previous year --- I commented, in an almost philosophical way, on the distance between theory (the "how it should be") and practice (the "how it is") in such an area of study. I discussed topics such as "the lack of formal rigor (that) should clearly delimit the extent and meaning of the term," "the lack of a common understanding of which factors should prevail in the construction of this elementary requirement," and the lack of "absolute limits between strabismus and normality." I analyzed the conceptual aspects of the characterization of strabismus (its definition and elements, the precision of measurements and their limits, the referential coordinate systems, the primary position of the eye, the ocular movements in space, the "normal" positional asymmetries of the eye in space), and the operational aspects of these characterizations (the difficulties for the practical establishment of the so-called "primary position of gaze"—a concept that I assume to be only theoretical—and the methods of quantifying strabismus measurements). I ended up with ten "conclusions" for the pragmatic coexistence with such "epistemic dissonances."

Evidently, being able to monitor how the muscles are positioned over the eye during eye movements (by nuclear magnetic resonance imaging); directly injecting into them substances that reduce or increase their actions; stimulating or depressing neuronal (motor and sensory) functions; and develop further knowledge from experimental work, will all bring enormous progress to the *practice* of strabismus diagnosis and treatment. But, concomitantly, for "strabismology" to become more and more a *science*, its basic concepts must be revisited. For my part, this is what (I think) I can do: in one of my most recent articles[145], I addressed, in depth, one of these "dissonances" which were already previously referred[146] — the imprecision of our basic unit of angular measurements (the prism-diopter) — to make it more tolerable.

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